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THE CRITICAL FLICKER FREQUENCY DURING
PROLONGED MONOCULAR DEPRIVATION

John P. Zubek, et al

Manitoba University
Winnipeg, Canada

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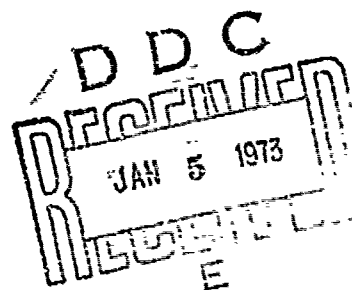
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Depression and Later Enhancement of the Critical Flicker Frequency during Prolonged Monocular Deprivation

John P. Zubek and M. Bross

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Depression and Later Enhancement of the Critical Flicker Frequency during Prolonged Monocular Deprivation

Abstract: *One eye was visually deprived for 1 day, and the critical flicker frequency in the other eye was determined at the start of the deprivation period and then at intervals of 3, 6, 9, 15, and 24 hours. There was an initial depression in performance, followed by an enhancement effect. No significant changes in the critical flicker frequency were observed in the occluded eye at corresponding times; thus the depression-enhancement phenomenon is specific to the nonoccluded eye.*

In 1923, Allen (1) reported that 3 hours of monocular light deprivation produced a decrease in the critical flicker frequency (CFF) of the non-occluded eye, a result confirmed by Hollenberg (2). In contrast, we demonstrated (3) a negatively accelerating improvement in the CFF of the non-occluded eye during 1 week of monocular deprivation, the first measurement being taken 8 hours after deprivation was begun and the remainder at daily intervals. Furthermore, a sizable after-effect in this eye was still present 1

week after the removal of the black patch from the other eye. These two sets of results suggest that prolonged monocular deprivation may initially produce a depression of the CFF in the nonoccluded eye, and that the depression is followed by an enhancement effect of negatively accelerating magnitude. We report that this hypothesis was tested and confirmed.

Thirty male university students, all with normal vision, were divided into experimental and control groups, each containing 15 subjects. All subjects were required to live for 1 day in a large room (3.66 by 14.02 m), which was furnished with sofas, comfortable chairs, and study desks, and contained a radio, a television set, playing cards, and reading material. The mean ambient illumination, measured at desk height in eight different positions in the room, was 550 lu/m^2 . A washroom, a kitchenette, and sleeping quarters were adjacent to this furnished room. The subjects were confined to these apartment-like quarters in groups of three, two from one group and one from the other group. During the day spent in the room, each experimental subject wore a black patch over the dominant eye.

The CFF determinations were made before the patch was put in place and then at intervals of 3, 6, 9, 15, and 24 hours, the first measurement being made between 8:30 and 9:30 a.m. Subjects were permitted to sleep for approximately 8 hours after completion of the 15-hour test and were awakened 1 hour before the start of the 24-hour test (4). In order to control for possible effects of changes in blood sugar level on the CFF, each of the six determinations was made after the subject had eaten a meal or a snack that included a chocolate bar.

Before the CFF was measured at each test period, the nonoccluded (nondominant) eye of each experimental subject was dark-adapted for 15 minutes (the other eye was already covered by a patch). This duration was felt to be sufficient for testing with a small, centrally fixated visual target (4). The CFF was then taken from the nonoccluded eye only, since the presentation of a bright light to one eye can affect the CFF in the other eye (1, 2, 5). For each confined control subject, the CFF of the nondominant eye at each interval was determined after 15 minutes of binocular dark adaptation.

The stimulus consisted of a white

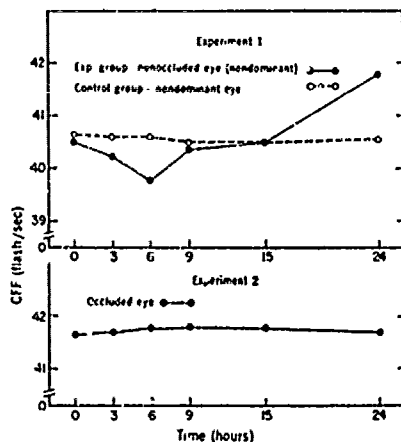


Fig. 1. Temporal changes in mean CFF values of subjects exposed to 24 hours of monocular deprivation. (Top) Measurements were taken on the nonoccluded (nondominant) eyes of the experimental subjects and on the nondominant eyes of control subjects. (Bottom) Measurements were taken on the occluded (dominant) eyes.

light, at an initial flicker frequency considerably higher than the fusion frequency. The light was presented monocularly by a cold cathode modulating lamp (Sylvania, type R1131c), mounted at the rear of a standard viewing chamber (Lafayette, model 1202C). The angle subtended by the centrally fixated stimulus was $2^\circ 10'$, a value assuring full foveal stimulation. The flicker-generating apparatus (Grason-Stadler, model E622) was set at a light to dark ratio of 0.50 and a lamp current

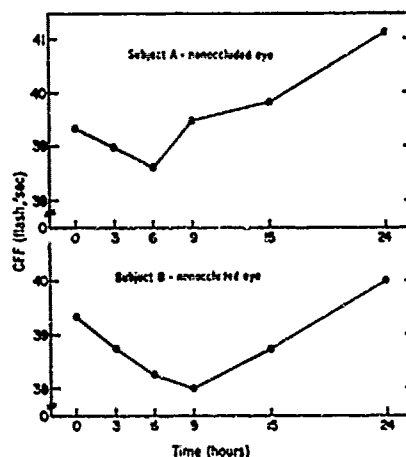


Fig. 2. CFF patterns in individual subjects. In subject A, the CFF for the nonoccluded eye is enhanced relatively early in the experiment. (Bottom) In subject B, the enhancement effect is seen only at the end of 24 hours of monocular deprivation.

reading of 22.6 ma. Eight trials, separated by 5-second intervals, were presented to the nondominant eye at each test period. The descending method of limits was used.

The CFF of the nonoccluded eyes shows an initial depression, as reported by Allen (1) and Hollenberg (2), a reversal toward the baseline level at 9 hours, and an enhancement effect at 24 hours (Fig. 1, top). On the other hand, the CFF of corresponding eyes of the confined controls does not change during the 24-hour session. An analysis of variance revealed a significant change over hours ($F = 21.82$, $P < .001$) and a significant interaction effect ($F = 20.29$, $P < .001$). A series of two-tailed *t*-tests comparing the relative performances of the two groups of subjects at the various time periods indicated that the decrease in the CFF at 3 and 6 hours, the reversal between 6 and 9 hours, and the increase at 24 hours were all statistically significant (P 's $< .01$).

An examination of the individual performance patterns of the 15 experimental subjects suggested the presence of two main types of "reactors" (Fig. 2). The first type, comprising a third of the sample, showed a prolonged period of depression with the enhancement effect appearing only at the 24-hour test, while the second type exhibited a relatively brief period of depression (at 3 and 6 hours only), and then an enhancement effect of progressively increasing magnitude. These two general response patterns may reflect possible differences in the degree of stress experienced by the experimental subjects resulting from the novelty of wearing an eye patch. Although no measures of stress or affect were made, we felt that subjects of the first type were somewhat more apprehensive and complained more often during the confinement period than did those showing the second type of response.

To determine whether a similar type of depression-enhancement phenomenon could be shown in the occluded eye, we did a second experiment. In each of nine subjects, the dominant eye was occluded for 1 day, and measurements were taken from this eye before the patch was put in place and then at intervals of 3, 6, 9, 15, and 24 hours. Before each test period, the nonoccluded eye was dark-adapted for 15 minutes, but it was not tested. (For the first measurement, both eyes were dark-

adapted since the black patch had not been applied.) There was no change in the CFF of the occluded eye at any time (Fig. 1, bottom). Thus, the depression-enhancement phenomenon is specific to the nonoccluded eye (4).

Although numerous variables affect the CFF, it is difficult to understand how any of them can account for both our present and earlier (5) results for the nonoccluded eye. The unusual time course, together with the persistence of the phenomenon for many days, suggests the disturbance of some interocular mechanism in the higher levels of the visual system. We believe that prolonged monocular deprivation may be producing changes in certain areas of the primary sensory system, changes similar to the denervation supersensitivity that occurs in the higher neural centers after partial surgical deafferentation at lower levels of the central nervous system (6). For example, Spiegel and Szekely (7) reported that lesions in the posteroventral nucleus of the thalamus (relay nucleus for touch) are followed, after an initial period of depression of the somesthetic cortex, by a hyperexcitability of this region. [More than a century ago, Hall (8) observed that "the first effect of injury done to the nervous system is a diminution of its functions, whilst the second or ulterior effect is the augmentation of these functions."] Occlusion of one eye, therefore, may be producing a state of temporary partial deafferentation of the visual system, a condition that is reflected behaviorally in the production of our CFF phenomenon. However, in contrast to surgically induced deafferentation, this deafferentation is functional, that is, it is produced by depriving the normal, intact organism of some of its accustomed visual experience.

This hypothesis is consistent with

Sharpless's (9) revision of the law of denervation (6), which has as its main thesis that supersensitivity results from prolonged disuse of neural pathways. Sharpless states, "Disuse may be the result of drugs, privation of sensory experience, or, most commonly, injury produced by severance of nervous pathways." Further, he says that supersensitivity is a compensatory process that occurs as a consequence of "a radical and sustained change in the level of input to an excitable structure." This explanation of disuse of neural pathways, with which we concur, has the merit of bringing together our results, the increased cutaneous sensitivity that occurs in human subjects after prolonged partial occlusion of the skin (10), and the various supersensitivity phenomena induced by surgery or by drugs. This explanation does not, however, adequately account for the presence of the CFF phenomenon in only one eye, nor does it indicate the specific neural locus of the interocular effect. Only future behavioral and electrophysiological research can provide satisfactory answers to these two problems.

Finally, our results are important in two general respects. First, they indicate that the monocular deprivation technique may provide a new method of attacking the complex problem of the physiological mechanisms underlying sensory isolation effects (11), an approach that can be used both in studies of humans and in electrophysiological studies in animals. Second, they suggest that many of the apparently contradictory results from isolation chamber studies (11), particularly those involving periods of 1 day or less and employing various sensory and perceptual-motor measures, may be accounted for by differences in the duration of experimental conditions. (The most commonly used periods have been 3, 9, 12,

and 24 hours.) As we have demonstrated, performance on the same measure may be either impaired, improved, or not affected, the specific effect being dependent upon the duration of deprivation. It has been assumed by most previous investigators in the sensory deprivation area that this experimental variable is probably not too important and therefore can be ignored. This assumption is no longer valid.

JOHN P. ZUBEK, M. BROSS
Department of Psychology, University
of Manitoba, Winnipeg, Canada

References and Notes

1. F. Allen, *J. Opt. Soc. Amer.* 7, 583 (1923).
2. M. S. Hollenberg, *ibid.* 8, 713 (1924).
3. M. Bross and J. P. Zubek, *Can. J. Psychol.* 26, 42 (1972).
4. The depression-enhancement phenomenon is also present in the dominant nonoccluded eye; the temporal pattern is not affected by the use of 30 minutes of dark adaptation at each of the test periods; and, finally, it can be obtained even when the subject is awake during the entire 24-hour period. Further evidence that sleep was not a confounding variable, particularly in producing the sizable enhancement effect at 24 hours (Fig. 1, top), is provided by the observation that most subjects in experiment 1 showed an increased CFF at least 6 hours before going to bed and that this effect then increased in magnitude with time.
5. C. S. Sherrington, *The Integrative Action of the Nervous System* (Yale Univ. Press, New Haven, Conn., 1906).
6. W. B. Cannon and A. Rosenblueth, *The Supersensitivity of Denervated Structures* (Macmillan, New York, 1949); G. W. Stavraky, *Supersensitivity following Lesions of the Nervous System* (Univ. of Toronto Press, Toronto, 1961).
7. E. Spiegel and E. G. Szekely, *Electroencephalogr. Clin. Neurophysiol.* 7, 375 (1955).
8. M. Hall, quoted by G. W. Stavraky, *Supersensitivity following Lesions of the Nervous System* (Univ. of Toronto Press, Toronto, 1961), p. 3.
9. S. K. Sharpless, *Annu. Rev. Physiol.* 26, 357 (1964).
10. M. Aftanas and J. P. Zubek, *Percept. Mot. Skills* 16, 565 (1963); *ibid.* 17, 867 (1963); *ibid.* 18, 437 (1964); W. Heron and G. R. Morrison, unpublished manuscript.
11. J. P. Zubek, Ed., *Sensory Deprivation: Fifteen Years of Research* (Appleton-Century-Crofts, New York, 1969).
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